FACTORS AFFECTING VISUAL OUTCOME AND PROGRESION OF DIABETIC RETINOPATHY AFTER CATARACT SURGERY IN TYPE II DIABETIC PATIENTS

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CERTIFICATE

This is to certify that this dissertation titled "**FACTORS AFFECTING VISUAL OUTCOME AND PROGRESION OF DIABETIC RETINOPATHY AFTER CATARACT SURGERY IN TYPE II DIABETIC PATIENTS** " is the original and bonafide work done by **Dr. Ramya .R** under our guidance and supervision at the Government Stanley Medical College & Hospital, Chennai – 600 001, during the tenure of her course in M.S. Ophthalmology from June - 2008 to April-2011 held under the regulation of the Tamilnadu Dr. M.G.R. Medical University, Guindy, Chennai - 600032.

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CONTENTS

INTRODUCTION

Diabetes mellitus is a major medical problem throughout the world. Diabetes causes an array of long-term systemic complications, which have considerable impact on both the patient and the society because it typically affects individuals in their most productive years. Ophthalmic complications of diabetes include corneal abnormalities, glaucoma, iris neovascularization, cataracts, and neuropathies. However, the most common and potentially most blinding of these complications is diabetic retinopathy. Cataract occurs more frequently and at an earlier age in diabetic than in non-diabetic patients. Cataract surgery in diabetic patients may become necessary, not only to improve vision but also to allow assessment and treatment of diabetic retinopathy. Compared to non-diabetic patients, visual outcome after cataract surgery was reported to be worse in diabetic patients - especially in those with diabetic retinopathy. Some investigators also found an increased progression of retinopathy and a higher incidence of macular oedema after cataract surgery but others did not .Most of these studies were retrospective, and either different operation techniques - such as ICCE or ECCE, phacoemulsification (in a few recent studies), or different patient populations such as diabetic patients without retinopathy and with retinopathy of various degrees – may explain the different outcomes. Surgical outcome in patients without diabetic retinopathy is comparable to non-diabetic patients, and the outcome of patients with retinopathy appears to depend on the degree of retinopathy at the time of

surgery .In general, patients with mild non-proliferative retinopathy without laser indication have been proposed to have a good prognosis. Patients with mild-to-moderate diabetic retinopathy also represent a large proportion of patients with diabetic retinopathy. We therefore planned to valuate the visual outcome, the progression of retinopathy, and the incidence of macular oedema 1 year after small incision cataract surgery and intraocular lens implantation in a homogenous group of patients with mild-to-moderate diabetic retinopathy without clinically significant macular oedema at baseline. Progression of retinopathy and incidence of macular edema were compared with the non-operated fellow eyes.

PATHOPHYSIOLOGY OF DIABETIC RETINOPATHY

The exact mechanism by which diabetes causes retinopathy remains unclear, but several theories have been postulated to explain the typical course and history of the disease.

Platelets and blood viscosity

The variety of hematologic abnormalities seen in diabetes, such as increased erythrocyte aggregation, decreased red blood cell deformability, increased platelet aggregation, and adhesion, predispose to sluggish circulation, endothelial damage, and focal capillary occusion. This leads to retinal ischemia, which, in turn, contributes to the development of diabetic retinopathy.

Aldose reductase and vasoproliferative factors

Fundamentally, diabetes mellitus causes abnormal glucose metabolism as a result of decreased levels or activity of insulin. Increased levels of blood glucose are thought to have a structural and physiologic effect on retinal capillaries causing them to be both functionally and anatomically incompetent.

A persistent increase in blood glucose levels shunts excess glucose into the aldose reductase pathway in certain tissues, which converts sugars into alcohol (e.g., glucose into sorbitol, galactose to dulcitol). Intramural pericytes of retinal capillaries seem to be affected by this increased level of sorbitol,

eventually leading to the loss of its primary function (i.e., auto regulation of retinal capillaries).

Loss of function of pericytes results in weakness and eventual saccular out pouching of capillary walls. These microaneurysms are the earliest detectable signs of diabetic retinopathy.

Increased permeability of these vessels results in leakage of fluid and proteinaeous material, which clinically appears as retinal thickening and exudaes. If the swelling and exudation would happen to involve the macula, a diminution in central vision may be experienced. Macular edema is the most common cause of vision loss in patients with nonproliferative diabetic retinopathy.

Another theory to explain the development of macular edema deals with the increased levels of diacylglycerol from the shunting of excess glucose. This is thought to activate protein kinase C, which, in turn, affects retinal blood dynamics, especially permeability and flow, leading to fluid leakage and retinal thickening.

As the disease progresses, eventual closure of the retinal capillaries occurs, leading to hypoxia. Infarction of the nerve fiber layer leads to the formation of cotton-wool spots with associated stasis in axoplasmic flow.

More extensive retinal hypoxia triggers compensatory mechanisms within the eye to provide enough oxygen to tissues. Venous caliber

abnormalities, such as venous beading, loops, and dilation, signify increasing hypoxia and almost always are seen bordering the areas of capillary nonperfusion. Intraretinal microvascular abnormalities represent either new vessel growth or remodeling of preexisting vessels through endothelial cell proliferation within the retinal tissues to act as shunts through areas of nonperfusion.

Further increase in retinal ischemia trigger the production of vasoproliferative factors that stimulate new vessel formation. Neovascularization most commonly is observed at the borders of perfused and nonperfused retina and most commonly occur along the vascular arcades and at the optic nerve head. The new vessels break through and grow along the surface of the retina and into the scaffold of the posterior hyaloid face. These delicate vessels are disrupted easily by vitreous traction, which leads to hemorrhage into the vitreous cavity or the preretinal space.

These new blood vessels initially are associated with a small amount of fibroglial tissue formation. However, as the density of the neovascular frond increases, so does the degree of fibrous tissue formation. In later stages, the vessels may regress leaving only networks of avascular fibrous tissue adherent to both the retina and the posterior hyaloids face. As the vitreous contracts, it may exert tractional forces on the retina via these fibroglial connections. Traction may cause retinal edema, retinal heterotropia, and both tractional retinal detachments and retinal tear formation with subsequent detachment.

EPIDEMIOLOGY OF DIABETIC RETINOPATHY

Frequency

Diabetic retinopathy is emerging as one of the important causes of blindness in both developing and developed countries. The World Health Organization has estimated that, the number of adults with diabetes in the world would increase alarmingly: from 135million in 1995 to 300 million in 2025.

In India, this increase is expected to be the greatest; nearly 195% from 18 million in 1995 to 54 million in 2025.

Studies done by the Indian council of medical research in the early 1970's had shown the prevalence of diabetes in India to be 2.5% in the urban population and 1.5% in the rural population. However recent reports have shown the prevalence to be in the range of 12 to 14% in the urban population. Of these patients with diabetes, over 20% are expected to be suffering from diabetic retinopathy. The prevalence of diabetes in the rural population is expected to be about 5%.

Race

An increased risk of diabetic retinopathy appears to exist in patients with Native American, Hispanic, and African American heritage.

Sex

Sex does not appear to have any affect on the development of diabetes or diabetic retinopathy.

Age

With increasing duration of diabetes, or with increasing age since the onset of diabetes, there is a higher risk of developing diabetic retinopathy and the complications of diabetic retinopathy, including diabetic macular edema or proliferative diabetic retinopathy.

CLINICAL ASPECTS OF DIABETIC RETINOPATHY

History

In the initial stages, patients are generally asymptomatic; however, in the more advanced stages of the disease, patients may experience symptoms, including blurred vision, distortion, or visual acuity loss.

Examination

Microaneurysms

- Earliest clinical sign of diabetic retinopathy
- Secondary to capillary wall outpouching due to pericyte loss
- Appear as small red dots in the superficial retinal layers
- Fibrin and red blood cell accumulation in the microaneurysm lumen
- Rupture produces blot hemorrhages
- May appear yellowish in time as endothelial cells proliferate and produce basement membrane

Dot and blot hemorrhages

- Occur as microaneurysms rupture in the deeper layers of the retina such as the inner nuclear and outer plexiform layers.
- Appear similar to microaneurysms if they are small; may need fluorescein angiography to distinguish between the two

Flame-shaped hemorrhages - Splinter hemorrhages that occur in the more superficial nerve fiber layer.

Retinal edema and hard exudates - Caused by the breakdown of the blood-retina barrier, allowing leakage of serum proteins, lipids, and protein from the vessels.

Cotton-wool spots

- Nerve fiber layer infarction from occlusion of precapillary arterioles
- Fluorescein angiography - No capillary perfusion
- Frequently bordered by microaneurysms and vascular hyperpermeability

Venous loops, venous beading

- Frequently adjacent to areas of nonperfusion
- Reflects increasing retinal ischemia
- Most significant predictor of progression to PDR

Intraretinal microvascular abnormalities

- Remodeled capillary beds without proliferative changes
- Collateral vessels that do not leak on fluorescein angiography
- Usually can be found on the borders of the nonperfused retina

CLASSIFICATION OF DIABETIC RETINOPATHY

Early Treatment Diabetic Retinopathy Study [ETDRS]

Levels of Diabetic Retinopathy
Nonproliferative Diabetic Retinopathy (NPDR)

A. Mild NPDR

- At least one microaneursym

B. Moderate NPDR

- Hemorrhages or microaneurysms (H/Ma),hard exudates.
- Soft exudates, Venous beading (VB), and intraretinal microvascular abnormalities (IRMAs)
- definitely present.

C. Severe NPDR

- H/ Ma in all 4 quadrants
- VB in 2 or more quardrants
- IRMA in at least 1 quadrant

D.Very Severe NPDR

- Any two or more of the following
- H/ Ma in all 4 quadrants
- VB in 2 or more quardrants
- IRMA in at least 1 quadrant

Proliferative Diabetic Retinopathy

E. Early PDR

- New vessels on the retina

F. High-Risk PDR

- Mild new vessels on the disc (NVD) with vitreous haemorrhage
- Moderate to severe NVD (1/4 to ½ disc area) with or without vitreous haemorrhage.
- Moderate neovascularisation elsewhere (1/2 disc area) with vitreous haemorrhage.

Clinically Significant Macular Edema (any ONE of the following)

- 1. Thickening of the retina located 500 μm or less from the center of the macula
- 2. Hard exudates at 500 μm or less from the center of the macula with thickening of the adjacent retina
- 3. A zone of retinal thickening, one disc area or larger in size, any portion of which is one disc diameter or less from the center of the macula.

International Clinical Diabetic Retinopathy (DR) Disease Severity Scale

Proposed Disease Severity Level	Findings Observable With Dilated Ophthalmoscopy
No apparent DR	No abnormalities
Mild nonproliferative DR	Microaneurysms only
Moderate nonproliferative DR	More than "mild" but less than "severe"
Severe nonproliferative DR	Any of the following: 20 or more intraretinal hemorrhages in 4 quadrants Definite venous beading in 2 or more quadrants Prominent IRMA in 1 or more quadrants and no Neovascularization
Proliferative DR	1 or more of the following: Definite neovascularization Preretinal or vitreous hemorrhage

International clinical classification of diabetic retinopathy

Severity of diabetic macular edema

2 major levels, with subcategories for diabetic macular edema

Proposed Classification	**Findings Observable Upon Dilated Ophthalmoscopy**
Diabetic Macular Edema Absent	No retinal thickening or hard exudates in posterior pole
Diabetic Macular Edema Present	Some retinal thickening or hard exudates in posterior pole

If diabetic macular edema is present, it can be categorized as follows:

Proposed Classification	**Findings Observable Upon Dilated Ophthalmoscopy**
Mild Diabetic Macular Edema	Some retinal thickening or hard exudates in posterior pole but distant from the macula
Moderate Diabetic Macular Edema	Retinal thickening or hard exudates approaching the center of the macula but not involving the center
Severe Diabetic Macular Edema	Retinal thickening or hard exudates involving the center of the macula.

Hard exudates are a sign of current or previous macular edema. Diabetic macular edema is defined as retinal thickening and this requires a 3-dimensional assessment that is best performed by a dilated examination using slit-lamp bio microscopy and/or stereo fundus photography**.**

Patients with CSME should be considered for laser surgery. Appropriate laser photocoagulation surgery reduces the risk of visual loss by more than 50%, compared with no treatment ever. Even in the presence of 20/20 or better vision, patients with CSME should be considered for laser surgery because substantial recovery of reduced visual acuity is relatively unusual following treatment. A minority of patients have improvement in vision. In a majority of cases, the goal of treatment with laser photocoagulation is to stabilize the visual acuity. When treatment is deferred, as may be desirable when the center of the macula is not involved or imminently threatened, patients should be observed closely (at least every 3 to 4 months) for progression.

The diagnosis of diabetic macular edema can be difficult. Macular edema is best evaluated by dilated examination using slit-lamp biomicroscopy and/or stereo fundus photography. An ophthalmologist who treats patients for this condition should be familiar with relevant studies and techniques as per the ETDRS.

Effective surgical treatment and retreatment protocols have been detailed. Preoperatively, the ophthalmologist should discuss with the patient

side effects and risks of treatment. The goal of treatment is to reduce the rate of visual loss or stabilize visual acuity.

Most patients require more than one treatment session (average: 3-4), separated by two to four months, for retinal thickening to resolve. **(4).**

RISK FACTORS FOR DIABETIC RETINOPATHY

Duration of the diabetes

In type II diabetes , the incidence of diabetic retinopathy increases with the duration of the disease. Of patients with type II diabetes, 23% have NPDR after 11-13 years, 41% have NPDR after 14-16 years, and 60% have NPDR after 16 years.

Glucose control

For patients with non–insulin–dependent diabetes mellitus, the American Diabetes Association has suggested that glycosylated hemoglobin levels of less than 7% (reflecting long-term glucose levels) should be the goal in all patients to prevent or slow down the onset of diabetes-related complications.

Renal disease, as evidenced by proteinuria and elevated blood urea nitrogen/creatinine levels, is an excellent predictor of the presence of retinopathy. This probably is due to the fact that both conditions are caused by DM-related microangiopathies such that the presence and severity of one reflects that of the other. Evidence suggests that aggressive treatment of the nephropathy may have a beneficial effect on the progression of diabetic retinopathy and neovascular glaucoma.

Systemic hypertension, in the setting of diabetic nephropathy, correlates well with the presence of retinopathy. Independently, hypertension also may

complicate diabetes in that it may result in hypertensive retinal vascular changes superimposed on the preexisting diabetic retinopathy, further compromising retinal blood flow.

Proper management of hyperlipidemia (elevated serum lipids) may result in less retinal vessel leakage and hard exudate formation. The reason behind this is unclear.

Pregnant women without any diabetic retinopathy run a 10% risk of developing NPDR during their pregnancy. Of those with preexisting NPDR, 4% progress to the proliferative type.

In the Wisconsin Epidemiological Study of Diabetic Retinopathy cataract was the most common cause of legal blindness in older- onset diabetes, and the second most common cause in diabetic patients with younger-onset disease.

PATHOGENESIS OF DIABETIC CATARACT

The enzyme aldose reductase catalyzes the reduction of glucose to sorbitol through the polyol pathway, a process linked to the development of diabetic cataract.

It has been shown that the intracellular accumulation of sorbitol leads to osmotic changes resulting in hydropic lens fibers that degenerate and form sugar cataracts .In the lens, sorbitol is produced faster than it is converted to fructose by the enzyme sorbitol dehydrogenase. In addition, the polar character of sorbitol prevents its intracellular removal through diffusion. The increased accumulation of sorbitol creates a hyperosmotic effect that results in an infusion of fluid to countervail the osmotic gradient. Animal studies have shown that the intracellular accumulation of polyols leads to a collapse and liquefaction of lens fibers, which ultimately results in the formation of lens opacities.

Furthermore, studies have shown that osmotic stress in the lens caused by sorbitol accumulation induces apoptosis in lens epithelial cells leading to the development of cataract.

A study performed by **Oishi et al.** investigated whether aldose reductase is linked to the development of adult diabetic cataracts. Levels of aldose reductase in red blood cells of patients under 60 years of age with a short duration of diabetes were positively correlated with the prevalence of posterior

subcapsular cataracts. A negative correlation has been shown in diabetic patients between the amount of aldose reductase in erythrocytes and the density of lens epithelial cells, which are known to be decreased in diabetics compared to nondiabetics suggesting a potential role of aldose reductase in this pathomechanism.

The polyol pathway has been described as the primary mediator of diabetes-induced oxidative stress in the lens .Osmotic stress caused by the accumulation of sorbitol induces stress in the endoplasmic reticulum the principal site of protein synthesis, ultimately leading to the generation of free radicals. There are numerous recent publications that describe oxidative stress damage to lens fibers by free radical scavengers in diabetics. However, there is no evidence that these free radicals initiate the process of cataract formation but rather accelerate and aggravate its development. Hydrogen peroxide is elevated in the aqueous humor of diabetics and induces the generation of hydroxyl radicals .Furthermore, increased glucose levels in the aqueous humor may induce glycation of lens proteins, a process resulting in the generation of superoxide radicals and in the formation of advanced glycation endproducts. By interaction with cell surface receptors such as receptor for advanced glycation endproducts in the epithelium of the lens further hydrogen peroxide is generated.

In addition to increased levels of free radicals, diabetic lenses show an impaired antioxidant capacity, increasing their susceptibility to oxidative stress.

The loss of antioxidants is exacerbated by glycation and inactivation of lens antioxidant enzymes like superoxide dismutases.

In conclusion, a variety of publications support the hypothesis that the initiating mechanism in diabetic cataract formation is the generation of polyols from glucose by aldose reductase which results in increased osmotic stress in the lens fibers leading to their swelling and rupture.

STUDIES REGARDING THE INCIDENCE OF DIABETIC CATARACT

Several clinical studies have shown that cataract development occurs more frequently and at an earlier age in diabetic compared to nondiabetic patients Data from the Framingham and other eye studies indicate a three to fourfold increased prevalence of cataract in patients with diabetes under the age of 65, and up to a twofold excess prevalence in patients above 65 .The risk is increased in patients with longer duration of diabetes and in those with poor metabolic control. A special type of cataract—known as snowflake cataract—is seen predominantly in young type 1 diabetic patients and tends to progress rapidly. Cataracts may be reversible in young diabetics with improvement in metabolic control. The most frequently seen type of cataract in diabetics is the age-related or senile variety, which tends to occur earlier and progresses more rapidly than in nondiabetics.

The Wisconsin Epidemiologic Study of Diabetic Retinopathy investigated the incidence of cataract extraction in people with diabetes. Furthermore, additional factors associated with higher risk of cataract surgery were determined. The 10-year cumulative incidence of cataract surgery was 8.3% in patients suffering from type 1 diabetes and 24.9% in those from type 2 diabetes. Predictors of cataract surgery included age, severity of diabetic retinopathy and proteinuria in type 1 diabetics whereas age and use of insulin were associated with increased risk in type 2 diabetics.

A follow-up examination of the Beaver Dam Eye Study cohort, consisting of 3684 participants 43 years of age and older, performed 5 years after the baseline evaluation showed an association between diabetes mellitus and cataract formation . In the study, the incidence and progression of cortical and posterior subcapsular cataract was associated with diabetes. In addition, increased levels of glycated hemoglobin were shown to be associated with an increased risk of nuclear and cortical cataracts.

In a further analysis of the Beaver Dam Eye study the prevalence of cataract development was studied in a population of 4926 adults . Diabetic patients were more likely to develop cortical lens opacities and showed a higher rate of previous cataract surgery than nondiabetics. The analysis of the data proved that longer duration of diabetes was associated with an increased frequency of cortical cataract as well as an increased frequency of cataract surgery.

The aim of the population-based cross-sectional Blue Mountains Eye Study was to examine the relationship between nuclear, cortical, and posterior subcapsular cataract in 3654 participants between the years 1992 to 1994. The study supported the previous findings of the harmful effects of diabetes on the lens. Posterior subcapsular cataract was shown to be statistically significantly associated with diabetes. However, in contrast to the Beaver Dam Eye Study, nuclear cataract showed a weak, not statistically significant, association after adjusting for other known cataract risk factors.

A population-based cohort study of 2335 people older than 49 years of age conducted in the Blue Mountains region of Australia investigated associations between diabetes and the 5-year incidence of cataract. The results of this longitudinal study conducted by the same group of investigators as the Blue Mountains Eye Study demonstrated a twofold higher 5-year incidence of cortical cataract in participants with impaired fasting glucose. Statistically significant associations were shown between incident posterior subcapsular cataract and the number of newly diagnosed diabetic patients.

The Visual Impairment Project evaluated risk factors for the development of cataracts in Australians. The study showed that diabetes mellitus was an independent risk factor for posterior subcapsular cataract when present for more than 5 years.

A goal of the Barbados Eye study was to evaluate the relationship between diabetes and lens opacities among 4314 black participants . The authors found that diabetes history (18% prevalence) was related to all lens changes, especially at younger ages.

CATARACT SURGERY IN DIABETIC PATIENTS

Approximately 10-15% of all patients who undergo cataract surgery are diabetics.

The 10-year cumulative incidence of cataract surgery in patients with diabetes in the Wisconsin Epidemiological Study of Diabetic Retinopathy was two to five times higher than in comparable non diabetic population Cataract in diabetic patients decreases the visual acuity and makes an adequate examination as well as photocoagulation of the retina more difficult or nearly impossible. Therefore, it is necessary and important to perform cataract surgery for visual rehabilitation and also for diagnostic and therapeutic reasons. This is also postulated in other studies.

Nevertheless, patients with diabetes are treated more cautiously. Some studies have shown that cataract surgery causes progression of retinopathy with new hemorrhages, exudates, and macular edema. This progression is also associated with a poor visual outcome in about 1 in 10 patients.

These results have not been confirmed in other studies, however . Recent studies have found that the principal determinant of the postoperative visual outcome appears to be the presence or absence of a clinically significant macular edema at the time of surgery.

Diabetics with mild to moderate diabetic retinopathy represent a high proportion of all patients with diabetic changes of the retina. For this reason, we evaluated visual outcome and disease progression after cataract surgery in eyes with no or mild to moderate diabetic retinopathy at baseline and 1 year after surgery.

There has been a recent shift in emphasis towards earlier cataract extraction in diabetics. Cataract surgery is advisable before lens opacity precludes detailed fundus examination.

While the overall outcomes of cataract surgery are excellent, patients with diabetes may have poorer vision outcomes than those without diabetes. Surgery may cause a rapid acceleration of retinopathy, induce rubeosis or lead to macular changes, such as macular edema or cystoid macular edema. The worst outcomes may occur in operated eyes with active proliferative retinopathy and/or preexisting macular edema.

In diabetics with or without evidence of diabetic retinopathy the blood-aqueous barrier is impaired leading to an increased risk of postoperative inflammation and development of a sight-threatening macular edema, a process that is exacerbated by cataract surgery . Factors that influence the amount of postoperative inflammation and the incidence of clinical and angiographic cystoid macular edema are duration of surgery, wound size and posterior capsular rupture or vitreous loss.

An analysis of Medicare beneficiaries from the years 1997 through 2001 revealed that the rate of cystoid macular edema diagnosis after cataract surgery was statistically significantly higher in diabetic patients than in nondiabetics.

Several clinical studies investigated the role of cataract surgery on the progression of diabetic retinopathy.

One year after cataract surgery, the progression rate of diabetic retinopathy ranges between 21% and 32% **Borrillo et al.** reported a progression rate of 25% after a follow-up period of 6 months .A retrospective review of 150 eyes of 119 diabetic patients undergoing cataract surgery showed a similar progression of diabetic retinopathy in 25% of cases within the follow-up period of 6–10 months A prospective study evaluating the onset or worsening of macula edema at 6 months following cataract surgery in patients with mild or moderate nonproliferative diabetic retinopathy reported an incidence of 29% (30 of 104 eyes) of macula edema based on angiographic data. **Krepler et al.** investigated 42 patients undergoing cataract surgery and reported a progression of diabetic retinopathy of 12% in operated versus 10.8% in nonoperated eyes during the follow-up of 12 months . During the same follow-up period of 12 months, **Squirrell et al**. showed that out of 50 patients with type 2 diabetes undergoing unilateral cataract surgery 20% of the operated eye and 16% of the nonoperated had a progression of diabetic retinopathy. Liao and Ku found in a retrospective study that out of 19 eyes with preoperative

mild to moderate nonproliferative diabetic retinopathy 11 eyes (57.9%) showed progression of diabetic retinopathy 1 year after surgery, while 12 eyes (63.2%) had progressed 3 years postoperatively. The progression rates were statistically significant when compared to eyes without preoperative retinopathy.

A recently published prospective study evaluated eyes from 50 diabetic patients with and without retinopathy after cataract surgery by optical coherence tomography. The authors reported an incidence of 22% for macula edema following cataract surgery (11 of 50 eyes) while macula edema did not occur in eyes without retinopathy. When only eyes with confirmed diabetic retinopathy were evaluated, the incidence for postoperative macula edema and cystoid abnormalities increased to 42% (11 of 26 eyes).

Minimal changes from baseline values in center point thickness were observed in eyes with no retinopathy.

Eyes with moderate nonproliferative diabetic retinopathy or proliferative diabetic retinopathy developed an increase from baseline of 145microns and 131microns at 1 month and 3 month, respectively. The difference in retinal thickening between the 2 groups at 1 and 3 months was statistically significant and among patients with retinopathy inversely correlated with visual acuity improvements**.(13)**

Influence of diabetes on operative risk and wound healing

The time worn aphorism that postoperative morbidity is higher in persons with diabetes is not supported by clinical studies. The conventional postulate that optimal perioperative glycemic control enhances the chances of successful wound healing in diabetes is supported only by experimental studies in animal models of diabetes. These studies implicate hyperglycemia and insulin deficiency as factors contributing to impaired wound healing. Deficient formation of granulation tissue and collagen, poor tensile strength of deep surgical wounds and deficient capillary in growth into the wound have been demonstrated. **(8)**

Preoperative evaluation

In planning for surgery, attention should be directed towards ensuring that the general physical condition of the patient is as good as possible. With the changes imposed by shorter hospital stays, the physician has little time for careful study and correction of related medical problems before surgery.

The importance of pre- admission out-patient assessment cannot be over emphasized as it allows for identification and treatment of potential complicating conditions.

Assessment of cardiac risk

Mortality due cardiovascular disease increases sharply with age and duration of diabetes. Assessment of cardiac risk is a major focus during the pre-operative evaluation of the patient with diabetes. Particular attention should be paid to the history of previous cardiac disease and current cardiac symptoms. Surgery should be postponed for 3 to 6 months in patients with the history of recent myocardial infarction.

Assessment of renal disease

Renal disease is common in patients with long standing diabetes. Caution should be exercised in the use of iodine containing angiographic fluids and contrast agents as these are well recognized nephrotoxins in patients with significant renal insufficiency. All patients undergoing such studies should be kept well hydrated with the additional use of furosemide and mannitol, if needed, to prevent acute renal damage.

Treatment of hypertension

Hypertension is a common accompaniment of diabetes. All reasonable efforts should be made to bring the blood pressure under control before surgery. As a general rule, anti-hypertensive medications are given on schedule on the day of surgery.

Diabetes treatment during and after surgery

Management is considered satisfactory when the blood glucose levels in the peri-operative period range between 120-180 mg%. When peri-operative glucose values are less than 100%, careful vigilance is necessary to avoid hypoglycemia. The wide spread availability of capillary blood glucose monitoring has greatly enhanced our ability to control patient's blood glucose level during the peri-operative period.

Type 1 diabetes

Patients with this type of diabetes are a heterogenous group who require individualization of their treatment plan. Knowledge of a patient's previous glycemic control, best determined by the glycosylated haemoglobin (HbA1C) or fructosamine test, can be of help in determing pre-operative insulin requirement. Alterations in nutrition, variable glucose infusion rates, surgical stress and post operative pain and immobility are factors that influence post operative insulin requirement. For these reasons, insulin is often required during peri-operative period by the patient with poorly controlled Type II diabetes who is receiving oral agent.

Type 2 diabetes

Surgical patients receiving oral hypoglycemic agents fall into three categories:

i) those with good glycemic control-for this group, it is usually sufficient to administer the oral agents on the morning of surgery. Long acting agents like chlorpropamide should be stopped 24 hrs before surgery in favour of a shorter acting sulphonyl urea.

ii) those with poor glycemic control that will improve when infection clears or steroid therapy is discontinued- this group require insulin therapy temporarily during the peri-operative period

iii) those who actually require insulin therapy with oral agents having failed to control hyperglycemia- patients in this group have an absolute need for insulin therapy.

Dietary management

Individualized dietary prescriptions are recommended for the hospitalized adult patients with diabetes. Hospitilization is an ideal opportunity to obtain a nutritional assessment and to assist the patient in developing an appropriate dietary plan. Dietary management should be based on a sound nutritional assessment of the patient's caloric needs. **(8)**

AIM

To study the effect of cataract surgery and other factors on the visual outcome and progression of diabetic retinopathy using the nonoperated contralateral eye as the control.

MATERIALS AND METHODS

Monocular cataract surgery was performed in 100 patients who had the same degree of retinopathy in both eyes preoperatively. Patients were assigned to 1 of 2 groups as follows: **Group A**, progression of retinopathy in the operated eye caused by cataract surgery; **Group B**, no progression of retinopathy bilaterally, comparable level of progression in both eyes, or more progression of retinopathy in the nonoperated eye than in the operated eye. The differences between the 2 groups in age, duration of and treatment methods for diabetes, renal function were compared. The 12 months follow-up included evaluation of the progression of retinopathy.

INCLUSION CRITERIA

1. Patients with type 2 diabetes mellitus with no or mild or moderate nonproliferative diabetic retinopathy without CSME.

2. Patients with immature cataract in the study eye and patients with either no cataract or immature cataract in the fellow eye

EXCLUSION CRITERIA

1. pseudophakic patients

2. patients having undergone previous laser treatment.

3. patients with severe NPDR ,PDR

4. patients with mature cataract

PROCEDURE

PREOPERATIVE EVALUATION

Systemic evaluation comprised of assessment of diabetic status and type of antidiabetic medication used. A detailed history of diabetic control over the past 5 years was elicited to know whether the patient's glycemic status was stable, fluctuating or not controlled previously.

The present glycemic control was assessed by referring all patients who were known diabetic or detected to be diabetic on routine examination, to the diabetology department and surgical fitness obtained before doing cataract surgery.

For patients with poor control of blood sugar levels, surgery was deferred until the diabetic status was stabilized. Assosciated conditions like hypertension,cardiac status and renal status were given special attention in all cases while taking history and necessary investigations, if warranted,were done before taking up the patient for surgery.

A complete ocular examination was performed preoperatively and at the final checkup. This examination included the determination of best corrected visual acuity using Snellen charts, applanation tonometry, slitlamp examination, and retinal biomicroscopy at the slitlamp with a 90D lens. Fundus examinations were also performed by direct and indirect ophthalmoscopy. Diabetic retinopathy was graded accordingly.

Keratometry and A scan were done and intra ocular lens power calculated using SRK II formula.

SURGERY

All patients were operated only by a single senior surgeon.

Routine SICS with PCIOL was planned in these patients.

Lid preparation was done with betadine solution.

Bridle suture was applied

Fornix based peritomy was done.

Incision was made about 2mm from the limbus with 11 knife .Scleral tunnel incision was made with crescent knife.

Entry into anterior chamber was made with the keratome and viscoelastic material was injected into the chamber.

Continuous curvilinear capsulorrhexis was performed.Can opener capsulotomy was done in those patients in whom capsulorhexis could not be performed.

Hydrodissection was performed.

Nucleus was rotated and brought to the anterior chamber and delivered out using sandwich technique.

Cortical aspiration was done using a 23G simcoe cannula with the infusion of Ringer Lactate solution.

A complete cortical aspiration was ensured by appreciation of fundal glow.

Viscoelastic material was injected and capsular bag distended .

The integrity of capsular bag was ensured and then rigid PCIOL was inserted in the bag.

The cortical remnants and viscoelastic material were aspirated and anterior chamber formed.

In the event of posterior capsular rent vitrectomy was performed and anterior chamber IOL was inserted.

Injection subconjunctival gentamycin 10 mg and dexamethasone 0.5mg was injected at the end and dressing applied.

All patients were treated with systemic antibiotics and anti-inflammatory drugs.

Topical short acting cycloplegic and antibiotic steroid preparation was applied.

The patient was discharged on the 1st postoperative day if there were no immediate postoperative complications after doing trial vision, slit lamp examination, and fundus examination.

Patient showing evidence of maculopathic changes during this immediate postoperative period were considered to have macular lesions. They were followed up with FFA periodically and necessary intervention with laser photocoagulation done at appropriate time.

Postoperatvely all patients were followed up every week for 6 weeks and then at 12,24,48 weeks. Vision testing with pinhole and glasses slit lamp examination, fundus examination, and FFA if needed ,were done during the follow up visits and postoperative diabetic status monitored.

Blood sugar was done during follow up and postoperative diabetic status monitored. Refraction was done on the 6th postoperative week and glasses for residual error and near vision correction prescribed.

OUTCOME

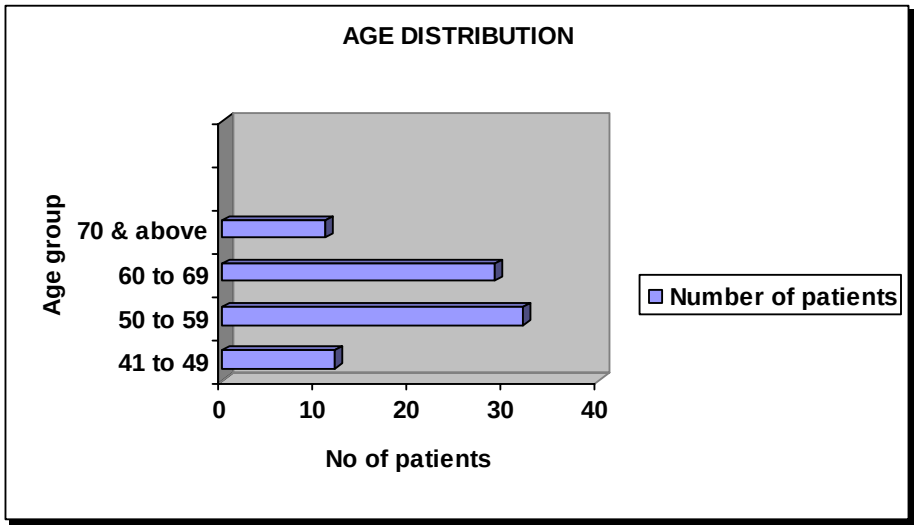
Outcome variables included changes of visual acuity, changes of retinopathy grading as compared to the nonoperated fellow eye, and incidence of macular edema.

ANALYSIS

AGE DISTRIBUTION

Table 1

Age, Years	Number of Patients
41 to49	12
50 to59	32
60 to 69	29
70&Above	11.



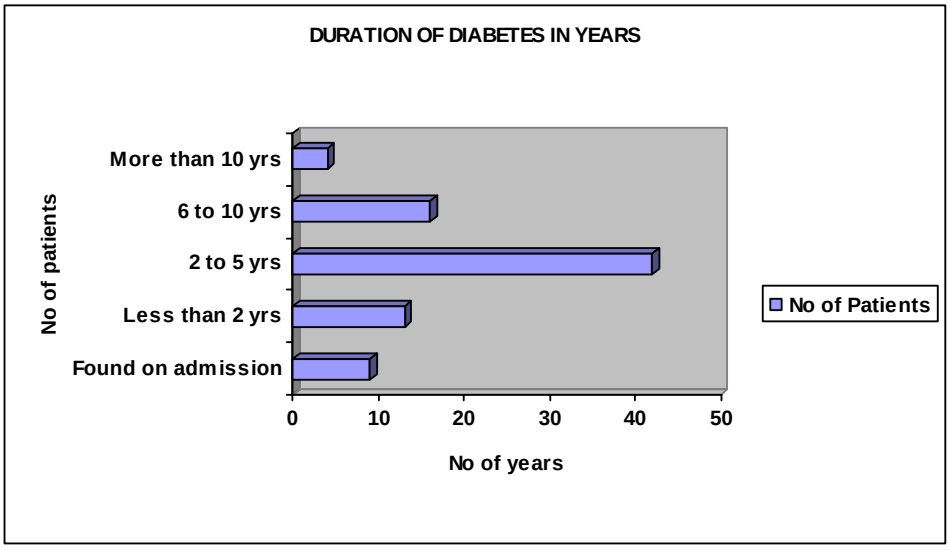
The age of the patients ranged from 41 yrs to 81 yrs, the mean age being 62 years. The maximum number of patients belong to the age group of 50 to 59.12 cases belong to the age group 41-49 which is in accordance to the studies that diabetes results in presenile type of cataract.

The mean age of surgery in a study by Pollack was 67.1 years.

DURATION OF THE DIABETES IN YEARS

Table 2

No of years	No of Patients
Found on admission	9
Less than 2 yrs	13
2 to 5 yrs	42
6 to 10 yrs	16
More than 10 yrs	4



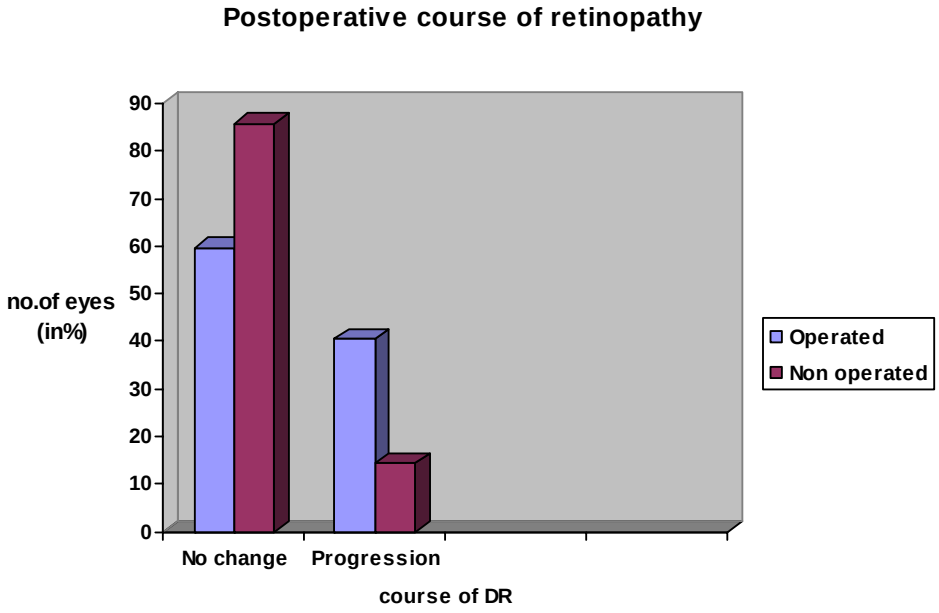
The majority of 42 patients with diabetes for 2 to 5 years had uncomplicated cataract. This shows that cataract in diabetes occurs even before the onset of retinopathy.

The mean duration of diabetes at the time of surgery was 5.5 years whereas Pollock showed the mean duration to be 10.1 years.

POSTOPERATIVE COURSE OF RETINOPATHY IN OPERATED AND NON OPERATED EYES

Table 3

EYE	NO CHANGE		PROGRESSION	
	NO. OF EYES	PERCENTAGE	NO. OF EYES	PERCENTAGE
OPERATED	50	59.6	34	40.4
NON OPERATED	72	85.7	12	14.3



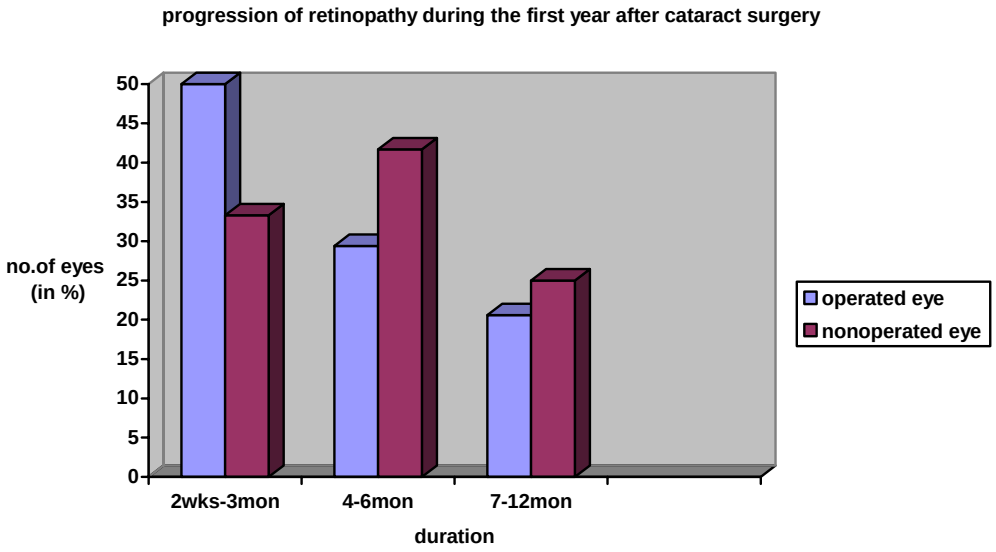
During the first postoperative year 50 of the operated eyes (59.5%) showed 'no change' in retinal status and 34 (40.4%) showed 'progression'of diabetic retinopathy. The corresponding findings in the control group were 72 eyes (85.7%) and 12 eyes (14.3%).

This was comparable to those of Pollock where 61.8% showed ‘no change’ and 38.2% progressed. The corresponding findings in the control group were 87.2% and 12.8%

PROGRESSION OF RETINOPATHY DURING THE FIRST YEAR AFTER CATARACT EXTRACTION IN OPERATED AND NON OPERATED EYES

Table 4

FOLLOW UP PERIOD	OPERATED EYE(n=34)		NON OPERATED EYE(n=12)	
	NO. OF EYES	PERCENTAGE	NO. OF EYES	PERCENTAGE
2weeks- 3 months	17	50	4	33.3
4-6 months	10	29.4	5	41.7
7-12 months	7	20.6	3	25



The operated eyes showed the highest percentage, that is, about 50% of operated patients showed progression of retinopathy from 2weeks to 3 months.

This was comparable to the study done by Pollock who showed 60% of progression from 2 weeks to 3 months.

REFERENCE STUDY

Progression of diabetic retinopathy during the first year after cataract extraction in operated eyes.

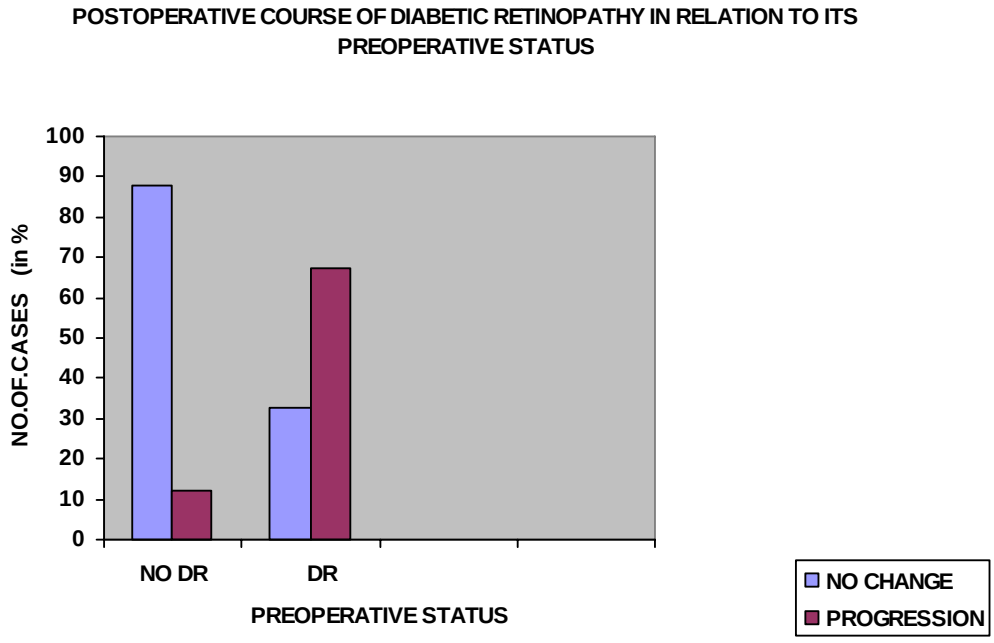
Follow-up period	No. of eyes progressed (in percentage)
2 weeks - 3 months	60
4 - 6 months	30
7 - 12 months	10

In this study there was maximum progression between the first 2 weeks and 3 months that is,60% and 30% of eyes progressed between 4 and 6 months.

POSTOPERATIVE COURSE OF DIABETIC RETINOPATHY IN RELATION TO ITS PREOPERATIVE STATUS IN THE OPERATED EYE

Table 5

PREOPERATIVE STATUS	NO.OF EYES(n=84)	NO CHANGE(n=50)	PROGRESSION(n=34)
NO DR	41	36 (87.8%)	5 (12.2%)
DR	43	14 (32.6%)	29 (67.4%)

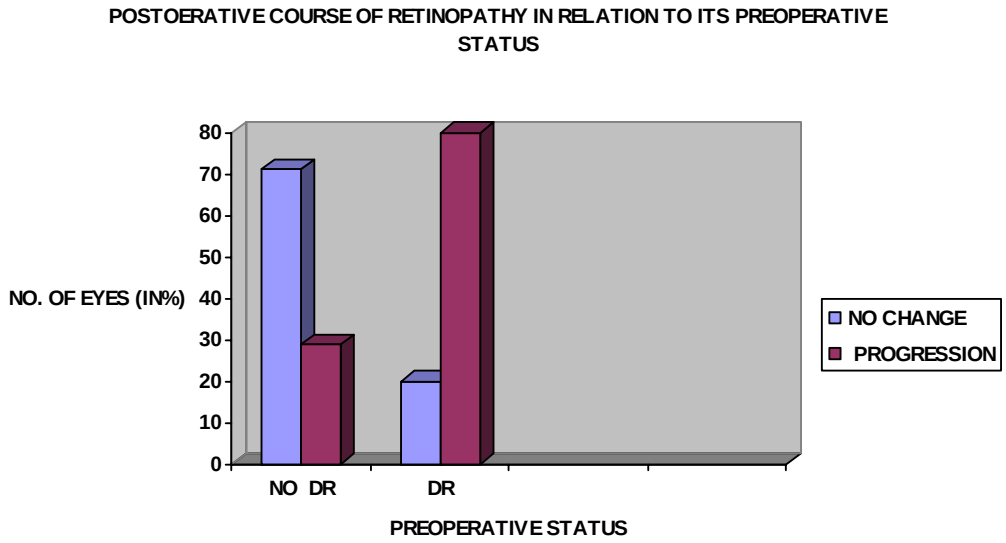


In eyes with no evidence of diabetic retinopathy preoperatively there was no progression of the retinopathy after cataract surgery in 87.8% of cases whereas 12.2% of cases progressed.

In eyes with evidence of retinopathy preoperatively there was no postoperative progression in 32.6% of cases whereas it progressed in 67.4% of cases.

In a study done by Pollock only 29% of eyes with no evidence of retinopathy preoperatively progressed whereas 80% of eyes with preoperative retinopathy progressed after surgery.

REFERENCE STUDY



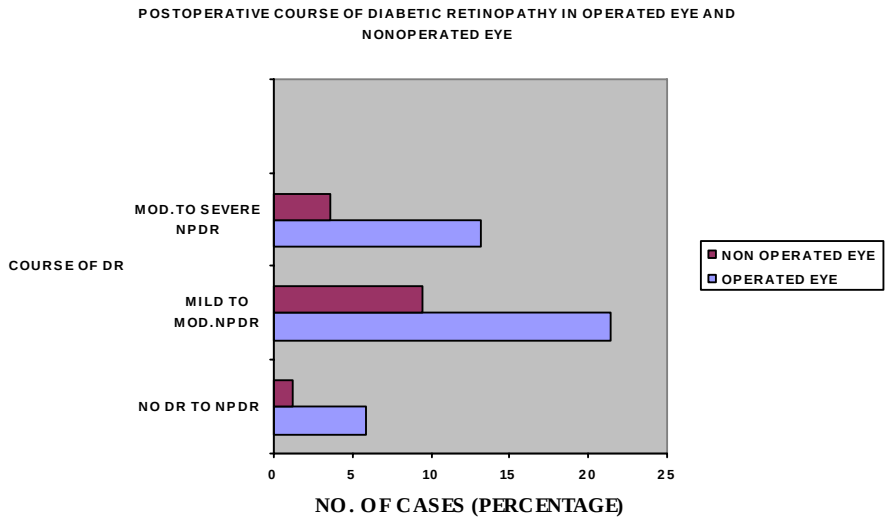
In 71% of eyes with no evidenc of retinopathy preoperatively there was no change even after surgery whereas only 29% of eyes progressed.

In eyes with evidence of retinopathy preoperatively there was no change in only 20% after surgery whereas about 80% of eyes progressed.

POSTOPERATIVE PATTERNS OF PROGRESSION OF DIABETIC RETINOPATHY IN OPERATED EYE AND NON OPERATED EYE

Table 6

COURSE OF DR	OPERATED EYE		NON OPERATED EYE	
	No.of cases	**Percentage**	**No.of cases**	**percentage**
No DR to NPDR	(5/84)	5.9	(1/84)	1.2
Mild to Mod.NPDR	(18/84)	21.4	(10/84)	9.5
Mod. To severe NPDR	(11/84)	13.1	(3/84)	3.6
Total	**(34/84)**	**40.4**	**(12/84)**	**14.3**



In eyes with no DR preoperatively there was progression to NPDR in 5.9 % of operated eyes and in 1.2% of the non operated eyes. In the operated eyes there was aggravation of mild NPDR to moderate NPDR in 21.4% of eyes whereas in the non operated eyes 9.5 % of eyes had progression from mild to moderate NPDR. In eyes. There was progression from moderate to severe NPDR in 13.1% of operated eyes the corresponding figures in the non operated eyes were 3.6%.

This was similar to the findings by Pollock where the maximum percentage of eyes (23.6%) showed aggravation of NPDR.

REFERENCE STUDY

POSTOPERATIVE PATTERNS OF PROGRESSION OF DIABETIC RETINOPATHY IN OPERATED EYE AND NON OPERATED EYE

COURSE OF RETINOPATHY	OPERATED (no. of eyes in %)	NON OPERATED (no. of eyes in %)
No DR to NPDR	9	4.3
NPDR to NPDR	23.6	7.1
No DR to NPDR NPDR to PDR	5.6	1.4
TOTAL	**38.2**	**12.8**

In this study there was postoperative progression in 38.2% of eyes among which 23.6% of eyes progressed from one stage of NPDR to a higher stage.

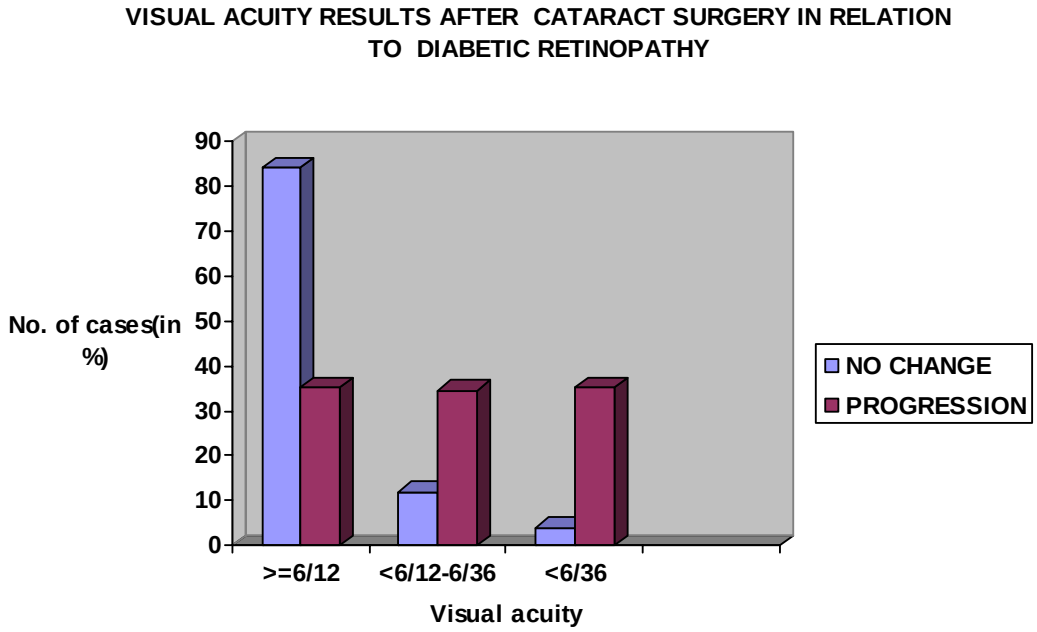
In the non operated eyes only 12.8% of eyes had postoperative progression of NPDR , 7.1% of which progressed from one form of NPDR to another form of NPDR.

VISUAL ACUITY RESULTS AFTER CATARACT SURGERY IN RELATION TO DIABETIC RETINOPATHY

Table 7

VISUAL ACUITY	NO CHANGE			PROGRESSION		
	NO DR (n=36)	DR (n=14)	TOTAL (n=50)	NO DR (n=5)	DR (n=29)	TOTAL (n=34)
>=6/12	32 (88.9%)	10 (71.4%)	42 (84%)	3 (60%)	9 (31%)	12 (35.3%)
<6/12-6/36	3 (8.3%)	3 (21.4%)	6 (12%)	1 (20%)	9 (31%)	10 (34.5%)
<6/36	1 (2.7%)	1 (7.1%)	2 (4%)	1 (20%)	11 (38%)	12 (35.3%)

In the no change group 84% of operated eyes had visual acuity more than 6/12 at the end of one year,12% had visual acuity in the range of 6/12 to 6/36 and 4% had visual acuity less than 6/36.In the eyes which had progression of retinopathy only 35.3% had visual acuity more than 6/12, of 34.5% in the range of less than 6/12 to 6/36 and 35.3% had less than 6/36 at the end of one year.



In eyes with no pre-existing retinopathy and no change in postoperative retinal status a visual acuity of at least 6/12 was achieved in 60% of the cases. By contrast, a relatively high proportion (38%) of eyes with pre-existing diabetic retinopathy and postoperative retinal deterioration had poor visual results of 6/36 or less.

REFERENCE STUDY

VISUAL ACUITY RESULTS AFTER CATARACT SURGERY IN RELATION TO DIABETIC RETINOPATHY

Visual Acuity	No change			Progression		
	NoDR (n=38)	DR (n=17)	Total (n=55)	NoDR(n=9)	DR(n=25)	Total(n=34)
>6/12	33 (87%)	12 (70%)	45 (82%)	6 (67%)	7 (28%)	13 (38%)
6/15-6/30	4 (10%)	3 (18%)	7 (13%)	2 (22%)	8 (32%)	10 (29%)
<6/30	1 (3%)	2 (12%)	3 (5%)	1 (11%)	10 (40%)	11 (32%)

In eyes with no evidence of diabetic retinopathy and no postoperative progression of retinopathy status about 87% of eyes had final visual acuity of more than 6/12 . 70% of eyes with preoperative retinopathy and no postoperative progression had more than 6/12 visual acuity.

In eyes with no postoperative progression 13% of eyes had visual acuity ranging from 6/15 to 6/30 and 5% had less than 6/30 vision postoperatively.

In the eyes which progressed after surgery 38% had more than 6/12 vision, 29% had 6/15- 6/30 and 32% had less than 6/30 vision.

INTRAOPERATIVE AND POSTOPERATIVE COMPLICATIONS

Among the intraoperative complications posterior capsular rent was observed in 4 eyes(4.8%) of cases which had an impact on the postoperative progression of retinopathy and the final visual status.

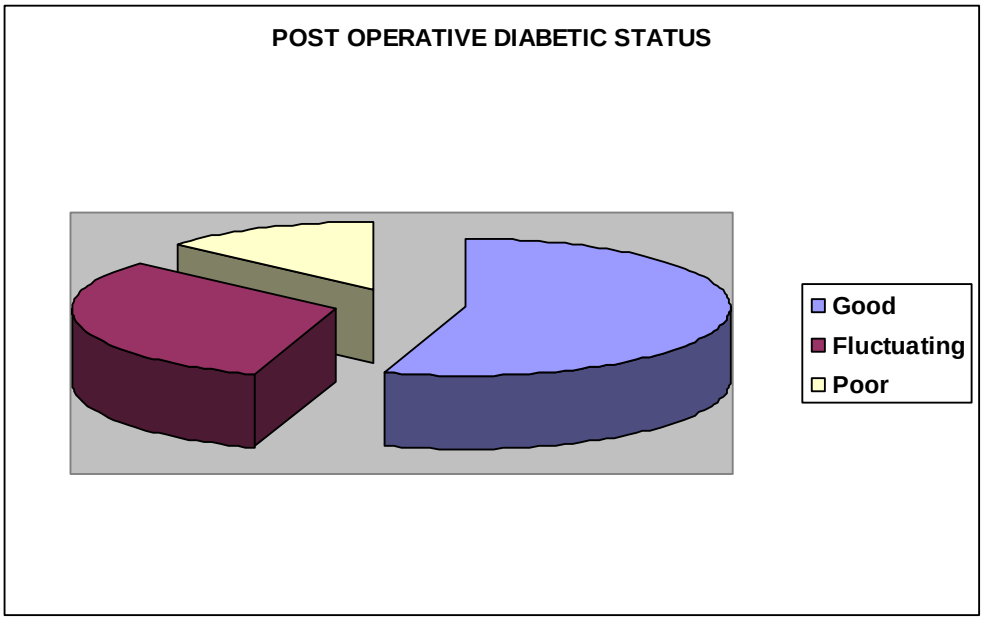
Of the early post operative complications, striate keratopathy was more frequent. It was observed in 16 eyes(19%). Iritis was observed in 10 eyes(12%).

Among the late post operative complications, CME occurred in 8 eyes(9.5%) and PCO occurred in 18 eyes(21.4%) of patients. Ruiz showed an incidence of PCO in 41.7% and Pasquier 30%.CME was observed in 32% of eyes in a study conducted by Pollack.

POST OPERATIVE DIABETIC STATUS

Table 8

Glycemic control	No of eyes	Percentage
Good	45	53.6
Fluctuating	26	31
Poor	13	15.4



In all these patients, good glycemic control was seen in 53.6% of patients. A fluctuating level was noticed in 31% of patients and a poor control in 15.4% of patients during the post operative period. CME and progression to retinopathy were more related to the fluctuating diabetic control during the post operative period.

COMPARISON OF CLINICAL FEATURES

Table 9

	NO CHANGE (n=50)	**PROGRESSION (n=34)**
Mean age, yr (range)	57.8 (45- 64)	61 (49-77)
Mean duration of DM, yr (range)	4.8	10.4
No. of. Patients (%)		
Male: female	23:27	16:18
Management of DM:		
Diet alone	1	1
Hypoglycemic agents	39 (78%)	21 (61.8%)
Insulin	10 (20%)	12 (35%)
Vascular disease:		
Hypertension	19	12
Cardiac	6	4
Other diseases:	2	3

There was no significant difference between the 'no change' and 'progression' group with regard to age, sex and systemic diseases.the mean duration of diabetes was 4.8 yrs in the no change group and 10.4yrs in the progression group. Regarding the management of diabetes 78% of patients in no change group were managed with oral hypoglycemic agents and 20% were managed with insulin whereas in the progression group 35% of patients were managed with insulin.

RESULTS

Of the 100 patients 46 were men and 54 were women.

7 patients lost to follow up. Of the 93 remaining patients cataract extraction was unilateral in 84 patients and bilateral in 9.Only the unilateral cases were included in the study.

The mean age at the time of surgery was 62 years (range 41 to 81 years) and the mean duration of diabetes mellitus was 5.5 years.

During the first postoperative year 50 of the operated eyes (59.6%) showed ‘no change’ in retinal status and 34 (40.4%) showed ‘progression’of diabetic retinopathy. The corresponding findings in the control group were 72 eyes (85.7%) and 12 eyes (14.3%) .

In eyes with no evidence of diabetic retinopathy preoperatively there was no progression of the retinopathy after cataract surgery in 87.8% of cases whereas 12.2% of cases progressed.

In eyes with evidence of retinopathy preoperatively there was no postoperative progression in 32.6% of cases whereas it progressed in 67.4% of cases.

The patterns of progression were similar in the operated and nonoperated eyes.

In eyes with no DR preoperatively there was progression to NPDR in 5.9 % of operated eyes and in 1.2% of the non operated eyes.

In the operated eyes there was aggravation of mild NPDR to moderate NPDR in 21.4% of eyes whereas in the non operated eyes 9.5% of eyes had progression from mild to moderate NPDR.

There was progression from moderate to severe NPDR in 13.1% of operated eyes and the corresponding figures in the non operated eyes were 3.6%.

In eyes with no pre-existing retinopathy and no change in postoperative retinal status a visual acuity of at least 6/12 was achieved in 88.9% of the cases. By contrast, a relatively high proportion (38%) of eyes with pre-existing diabetic retinopathy and postoperative retinal deterioration had poor visual results of 6/36 or less.

Among the intra operative complications posterior capsular rent was observed in 4.8% of cases. Of the early post operative complications, striate keratopathy was observed in 19% of patients. Iritis was observed in 12% of patients.

Among the late post operative complications, CME occurred on 9.5% and PCO occurred in 21.4% of patients.

Regarding the postoperative diabetic status good glycemic control was seen in 53.6% of patients. A fluctuating level was noticed in 31% of patients and a poor control in 15.4% of patients.

Among the operated patients there was no difference between the two subgroups with regard to sex distribution, mean age, or the presence of systemic disease. The only significant difference was related to the management and duration of diabetes mellitus.

The mean duration of diabetes was 4.8% in the ‘no change’ group and 10.4% in the ‘progression’ group Among the ‘no change’ group the diabetes was controlled by hypoglycemic agents in a greater number of patients(78%), whereas a greater number of patients whose retinopathy progressed needed insulin(35%) for management of diabetes

DISCUSSION

The aims of this study were

(1) to find out if the operated eyes of diabetic patients who undergo cataract surgery are more prone than non-operated eyes of the same patients to develop or to show progression of diabetic retinopathy;

(2) to identify the risk factors for postoperative progression of diabetic retinopathy; and

(3) to follow the course of the disease in this particular group of operated patients.

To achieve the last objective we attempted to eliminate any factors that might affect the natural course of the disease postoperatively.

Accordingly, we excluded from the study group eyes with any additional diseases such as glaucoma or macular diseases other than diabetes, or eyes treated by laser photocoagulation before or immediately after surgery for pre existing PDR or severe NPDR or CSME. Thus at the time of surgery the eyes in our study group presented only with No DR or mild to moderate NPDR. The differentiation between aphakic or pseudophakic cystoid macular oedema and diabetic cystoid macular oedema is a major problem. In this series 'progression' of diabetic retinopathy was defined only where worsening

of the characteristics typical for diabetic retinopathy were observed, regardless of whether foveal cystoid oedema was present or not.

Thus patients with cystoid macular oedema, but with no evidence of other characteristics of diabetic retinopathy, were included in the 'no change' group. Within 12 months of surgery diabetic retinopathy progressed in 40.4% of eyes that under-went cataract extraction as compared with only 14.3% in non- operated eyes over the same period (Table 3). The incidence of progression of diabetic retinopathy in our control group is in line with that reported by Pollock who studied the course of diabetic retinopathy over a one-year period in the general diabetic population aged 30 or older at the time of disease onset.

Since the pre-eminent risk variable for the occurrence of any diabetic retinopathy is thought to be the duration of the disease, this fact should be kept in mind when planning cataract surgery for diabetic patients.

Pollock et al also found 9% of operated eyes and 4.3% of non operated eyes having of new onset of retinopathy in patients who had been free of retinopathy at the start of the study.In our study only 5% of operated eyes and 1.2% of non operated eyes had new onset of retinopathy.

The commonest pattern of progression seen in our study group was worsening of pre-existing NPDR (Table 6) which was similar to the study by Pollock et al.

Pollock et al found that progression of retinopathy occurred nearly four times(7/27) as often in eyes with pre existing retinopathy than without it. In contrast we found that retinal deterioration occurred nearly six times (5/29) as often in eyes with preexisting diabetic retinopathy as without it (Table 5), thus pointing to the pre-existence of diabetic retinopathy as a possible risk factor for its postoperative progression.

The more frequent deterioration of diabetic retinopathy in our study group than in nonoperated eyes of this and of other series appears to support earlier suggestions that removal of the lens may result in a progression of diabetic retinopathy and/or development of rubeosis iridis, in contrast to the findings reported by Sebestyen, who observed similar progression of retinopathy in operated and nonoperated eyes. It is not yet clear how the removal of the lens may affect diabetic retinal changes in general.

However, with regard to one specific characteristic ofdiabetic retinopathy - the occurrence of endothelial proliferation and neovascularisation Williams et al showed that human lens extracts can inhibit endothelial proliferation. Therefore their removal may induce vascular alterations resulting in neovascularisation.

Furthermore they also demonstrated considerable inhibition of endothelial cell activity in extracts of bovine lens capsule, and suggested that the presence of an intact lens capsule may inhibit the development of iris neovascularisation.

Clinical experience seems to support these findings.

Aiello et al found that, following ICCE, patients with or without background retinopathy were at particularly high risk of developing vitreous haemorrhage, presumably reflecting progression of the disease to PDR. Alparl and Pollock et al observed deterioration of diabetic retinopathy in some diabetic patients following either ICCE or ECCE, with the least progression occurring in patients who underwent ECCE with IOL implantation in the capsular bag. Although clinical evidence suggests that ICCE may have a more deleterious effect than ECCE on the postoperative course of diabetic retinopathy, the precise role of the posterior lens capsule in reducing vascular complications after cataract surgery in diabetic patients requires further investigation.

Of the 34 eyes in our study that showed postoperative retinal deterioration none progressed to PDR but remained at the non-proliferative stage, sometimes with postoperative macular oedema, which affected the final visual results. The visual outcome following cataract surgery in our study (Table 7) was especially poor in patients with pre-existing diabetic

retinopathy who showed postoperative progression; it should however be noted that in the progression subgroup, good visual acuity was achieved in 60% of eyes with preoperative No DR and in 31% of eyes with preoperative NPDR.

The largest subgroup (88.9%) to achieve good visual results consisted of patients with no preexisting diabetic retinopathy and no change postoperatively.

The only significant difference in general clinical conditions between patients with no change in retinal status and patients with postoperative progression of diabetic retinopathy related to the management of diabetes and duration of diabetes : more patients in the 'progression' group than in the 'no change' group were managed by insulin and had longer duration of diabetes.

Other factors which influenced postoperative visual outcome were the incidence of cystoid macular edema (9.5%) and posterior capsular opacification (21.4%).

The results of this study clearly indicate that progression of diabetic retinopathy is not uncommon after cataract surgery, even when the technique employed is ECCE. Patients with diabetic retinopathy prior to surgery are at higher risk for progression. Accordingly these patients should be closely monitored postoperatively for early signs of progression of diabetic retinopathy and where necessary should be considered candidates for laser treatment. Diabetic patients scheduled for cataract extraction should be

informed that surgery may have an adverse influence on diabetic retinopathy and that this may affect the final visual outcome. The preoperative status of diabetic retinopathy may be a significant prognostic factor for the postoperative outcome.

CONCLUSION

Duration of diabetes plays a major role in the development of cataract. Cataract occurs more frequently in Type II diabetics of longer duration.

A good glycemic control during the perioperative period favourably influences the post operative visual outcome.

Cystoid macular edema and posterior capsular opacification were the most frequent postoperative complications.

Eyes with no evidence of diabetic retinopathy preoperatively showed a significantly higher incidence of no change than of progression in their postoperative retinal status. In eyes with preoperative non proliferative diabetic retinopathy the opposite was true: their postoperative retinal status showed a significantly higher incidence of progression than no change.

The incidence of the various patterns of progression observed one year after cataract surgery in the study eye with their corresponding incidence in the control eye showed higher percentage of progression of non proliferative diabetic retinopathy in both.

There is a significant increase in aggravation of preexisting diabetic eye status in patients with poor postoperative glycemic control and long standing diabetes and in those with posterior capsular rent.

Co existing systemic conditions do not play a major role in the visual recovery provided they are treated pre operatively.

Thus, cataract surgery in diabetic patients results in a good visual outcome.

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PROFORMA

1. Name :

2. Age :

3. Sex :

4. IP No. :

5. Occupation :

6. Address :

7. Complaints and duration :

8. Diabetic status of the patient

   a) Type of diabetes :

   b) Duration of diabetes :

   c) Mode of control of diabetes

      Diet and exercise :

      Oral hypoglycemic agents :

      Insulin :

9.Treatment for diabetic

Retinopathy (pre-op if any) :

10. Assosciated systemic illness :

11. Assosciated ocular illness :

12. Vision : RE LE

   a) With pinhole :

   b) Preliminary vision :

   c) subjective refraction :

13. Local examination : RE LE

Anterior segment

Lid :
Conjunctiva :
Cornea :
AC :
Iris :
Pupil :
Lens :

Fundus

Slit lamp biomicroscopy :

Indirect ophthalmoscopy :

14. Investigations

Intraocular pressure
(by applanation tonometer) :

Urine
Albumin :
Sugar :

Blood sugar
Fasting :

Postprandial :

Blood urea :

Serum creatinine :

Keratometry reading :

A scan :

15. Surgical notes and postoperative management

Date :

Anaesthesia :

RE/LE :

ACIOL/PCIOL :

Surgery :

16. Intraoperative complications

PC tear and its management :

17. Postoperative follow up

Weeks	1	2	3	4	5	6	12	24	36	48
Vision with pinhole										
Anterior segment										
Refraction										
Fundus										
PC thickening										
YAG capsulotomy										
FFA										
Laser photocoagulation										

18. Postoperative complications

Immediate

Weeks	1	2	3	4	5	6
Epithelial erosions						
Striate keratopathy						
Iritis						
Hyphaema						
Posterior synechiae						

Pupillary block							
Decentred lens							
Vitreous haemorrhage							
CME							
Endophthalmitis							

Late

Weeks	1	2	4	6	12	24	36	48
Bullous keratopathy								
High astigmatism								
Decentred lens								
Pigment deposits								
Delayed endophthalmitis								
PC thickening								
CME								
Worsening of DR								

19. Status of diabetic retinopathy

    Operated eye :

    Fellow eye :

20. Visual outcome :

ABBREVIATIONS

ACIOL	-	Anterior chamber intraocular lens
A Scan	-	Amplitude Scan
BA	-	Bronchial Asthma
CME	-	Cystoid macular edema
CSME	-	Clinically significant macular edema
DM	-	Diabetes mellitus
DR	-	Diabetic retinopathy
ECCE	-	Extracapsular cataract extraction
ETDRS	-	Early treatment for diabetic retinopathy study
FFA	-	Fundus flourescein angiography
H	-	Haemorrhage
HT	-	Hypertension
ICCE	-	Intracapsular cataract extraction
IHD	-	Ischemic Heart Disease
IRMA	-	Intra retinal microvascular abnormality
LE	-	Left Eye
Ma	-	Microanuerysm

No DR	-	No diabetic retinopathy
NPDR	-	Non proliferative diabetic retinopathy
NVD	-	Neovascularization disc
PC	-	Posterior Capsule
PCO	-	Posterior capsular opacification
PCIOL	-	Posterior chamber intraocular lens
RE	-	Right Eye
SICS	-	Small incision cataract surgery
SRK	-	Sanders Retzlaff Kraff
VB	-	Venous beading
YAG	-	Yttrium – Aluminium – Garnett

MASTER CHART

S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
1	Shanmugam	74	M	375832		RE	6/60	6/36	M0D	M0D	SEVERE	SEVERE	6/36
2	Kuppan	50	M	379245		LE	6/60	6/36	NO	NO	NO	NO	6/60
3	Lakshmanan	70	F	373539	HT	RE	6/36	6/12	M0D	M0D	SEVERE	M0D	6/60
4	George	60	M	358252		LE	6/24	6/9	NO	NO	NO	NO	6/6
5	Panchalai	60	F	17230		LE	6/24	6/9	M0D	M0D	M0D	M0D	6/12
6	Dillirani	68	F	17232	HT	RE	6/36	6/18	M0D	M0D	M0D	M0D	6/18
7	Mari	52	F	17740		LE	6/24	6/12	NO	NO	NO	NO	6/12
8	Shanthi	56	F	18521		LE	6/60	6/36	MILD	MILD	MILD	M0D	6/60
9	Rani	52	F	18774		LE	6/36	6/18	NO	NO	NO	NO	6/12
10	Pushpa	45	F	18589		RE	4/60	6/60	NO	NO	NO	NO	6/12
11	Vanasakshi	44	F	19813		LE	6/60	6/36	NO	NO	NO	NO	6/12
12	Kanmani	52	F	20876		RE	6/36	6/24	MILD	MILD	MILD	MILD	6/12
13	Selvakumar	52	M	20895		LE	5/60	6/60	MILD	MILD	MILD	MILD	6/12
14	Mani	70	M	20882	HT,IHD	RE	6/60	6/36	M0D	M0D	SEVERE	SEVERE	6/60

S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
15	Rukmani	55	F	21870	HT	LE	6/60	6/60	MILD	MILD	MILD	MILD	6/24
16	Krishnamoorthy	50	M	21861		LE	6/24	6/12	NO	NO	NO	NO	6/12
17	Manohari	55	F	21863		RE	6/60	6/36	MILD	MILD	M0D	MILD	6/18
18	Venkatesan	44	M	22543		LE	5/60	6/60	NO	NO	NO	NO	6/18
19	Selvaraj	45	M	22618		RE	6/36	6/18	NO	NO	NO	NO	6/12
20	Indirani	58	F	22168	HT	LE	6/36	6/18	MILD	MILD	MILD	MILD	6/12
21	Singaram	56	M	25281		RE	6/60	6/36	NO	NO	NO	NO	6/12
22	Swamy	60	M	26466		LE	6/60	6/36	MILD	MILD	M0D	M0D	6/18
23	Alamelu	38	F	19235		RE	6/36	6/12	NO	NO	NO	NO	6/9
24	Dhanam	48	F	8892		LE	6/24	6/9	NO	NO	NO	NO	6/6
25	Natarajan	67	M	27318		LE	6/24	6/9	MILD	MILD	M0D	M0D	6/24
26	Chandran	50	M	27913	HT,BA	RE	6/36	6/18	NO	NO	NO	NO	6/12
27	Manjula	33	F	28782		LE	6/24	6/12	NO	NO	NO	NO	6/12
28	Anwar	60	M	0853		LE	6/60	6/36	M0D	M0D	M0D	M0D	6/12
29	Rani	52	F	29264		LE	6/36	6/18	NO	NO	NO	NO	6/12

S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
30	Ajirabee	55	F	1396		RE	4/60	6/60	MILD	MILD	MILD	MILD	6/36
31	Subramanian	80	M	2211		LE	6/60	6/36	MILD	MILD	M0D	M0D	6/36
32	Nagammal	55	F	16609		RE	6/36	6/24	NO	NO	MILD	NO	6/12
33	Anandhan	66	M	3151		LE	5/60	6/60	M0D	M0D	M0D	SEVERE	6/60
34	Santhanalakshmi	70	F	3009	HT	RE	6/60	6/36	MILD	MILD	M0D	M0D	6/36
35	Vengammal	60	F	4220	HT	LE	6/60	6/60	MILD	MILD	MILD	M0D	6/12
36	Anjali	50	F	3886		LE	6/24	6/12	NO	NO	NO	NO	6/12
37	Vijayarani	50	F	4077		RE	6/60	6/36	MILD	MILD	M0D	M0D	6/24
38	Esther	44	F	5254		LE	5/60	6/60	NO	NO	NO	NO	6/12
39	Ethiraj	62	M	5136		RE	6/36	6/18	MILD	MILD	M0D	MILD	6/12
40	Thulasi	65	F	5756	HT	LE	6/36	6/18	MILD	MILD	MILD	MILD	6/12
41	Devagi	60	F	3242		RE	6/60	6/36	MILD	MILD	MILD	MILD	6/12
42	Lakshmi	60	F	5857		LE	6/60	6/36	MILD	MILD	MILD	M0D	6/12
43	Selvam	56	M	6107		RE	6/36	6/12	NO	NO	NO	NO	6/9
44	Padma	50	F	7192		LE	6/24	6/9	NO	NO	NO	NO	6/6

S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
45	Padmavathi	55	F	6878		LE	6/24	6/9	NO	NO	NO	NO	6/12
46	Rajeshwari	54	F	5868		RE	6/36	6/18	NO	NO	NO	NO	6/12
47	Arammal	60	F	6869		LE	6/24	6/12	NO	NO	NO	NO	6/12
48	Agustin	71	M	6732	HT	LE	6/60	6/36	M0D	M0D	SEVERE	SEVERE	6/60
49	Maasilamani	55	M	7719		LE	6/36	6/18	NO	NO	NO	NO	6/12
50	Ulaganathan	65	M	7749	HT	RE	4/60	6/60	M0D	M0D	SEVERE	M0D	6/60
51	Saraswathy	60	F	7746		LE	6/60	6/36	MILD	MILD	M0D	M0D	6/12
52	Rajamani	55	F	6336		RE	6/36	6/24	NO	NO	NO	NO	6/12
53	Ramamoorthy	73	M	8231	HT,IHD	LE	5/60	6/60	M0D	M0D	M0D	SEVERE	6/60
54	Arumugam	57	M	29603		RE	6/60	6/36	MILD	MILD	MILD	MILD	6/12
55	Gajendiran	74	M	32971	HT	LE	6/60	6/60	MILD	MILD	MILD	M0D	6/18
56	Kala	40	F	30397		LE	6/24	6/12	NO	NO	NO	NO	6/12
57	Panjali	65	F	31282		RE	6/60	6/36	MILD	MILD	M0D	M0D	6/12
58	Thesammal	67	F	31568	HT	LE	5/60	6/60	M0D	M0D	M0D	SEVERE	6/60
59	Jayashree	60	F	22137		RE	6/36	6/18	NO	NO	MILD	NO	6/60

S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
60	Kamatchi	58	F	43401		LE	6/36	6/18	NO	NO	NO	NO	6/12
61	Dhanapal	52	M	32423		RE	6/60	6/36	NO	NO	NO	NO	6/12
62	Kuppusamy	70	M	38291		LE	6/60	6/36	MILD	MILD	MILD	M0D	6/12
63	Fathima	42	F	24190		RE	6/36	6/12	NO	NO	NO	NO	6/9
64	Dharani	35	M	26337		LE	6/24	6/9	NO	NO	NO	NO	6/6
65	Navaneetham	45	M	26083		LE	6/24	6/9	NO	NO	NO	NO	6/12
66	Kothandam	50	M	27725	BA	RE	6/36	6/18	NO	NO	NO	NO	6/24
67	Lakshmi	45	F	28621		LE	6/24	6/12	NO	NO	NO	NO	6/12
68	Gangadharan	56	M	29489	HT	LE	6/60	6/36	M0D	M0D	M0D	M0D	6/60
69	Saraswathy	55	F	30242		LE	6/36	6/18	NO	NO	NO	NO	6/12
70	Govindaraj	67	M	31155	HT	RE	4/60	6/60	M0D	M0D	SEVERE	M0D	6/36
71	Soundararajan	66	M	31446		LE	6/60	6/36	MILD	MILD	M0D	M0D	6/60
72	Amaravathy	65	F	32340		RE	6/36	6/24	NO	NO	MILD	MILD	6/18
73	Selvaperumal	71	M	32310	HT,IHD	LE	5/60	6/60	M0D	M0D	M0D	SEVERE	6/60
74	Sivamugan	51	M	33027		RE	6/60	6/36	MILD	MILD	MILD	MILD	6/12

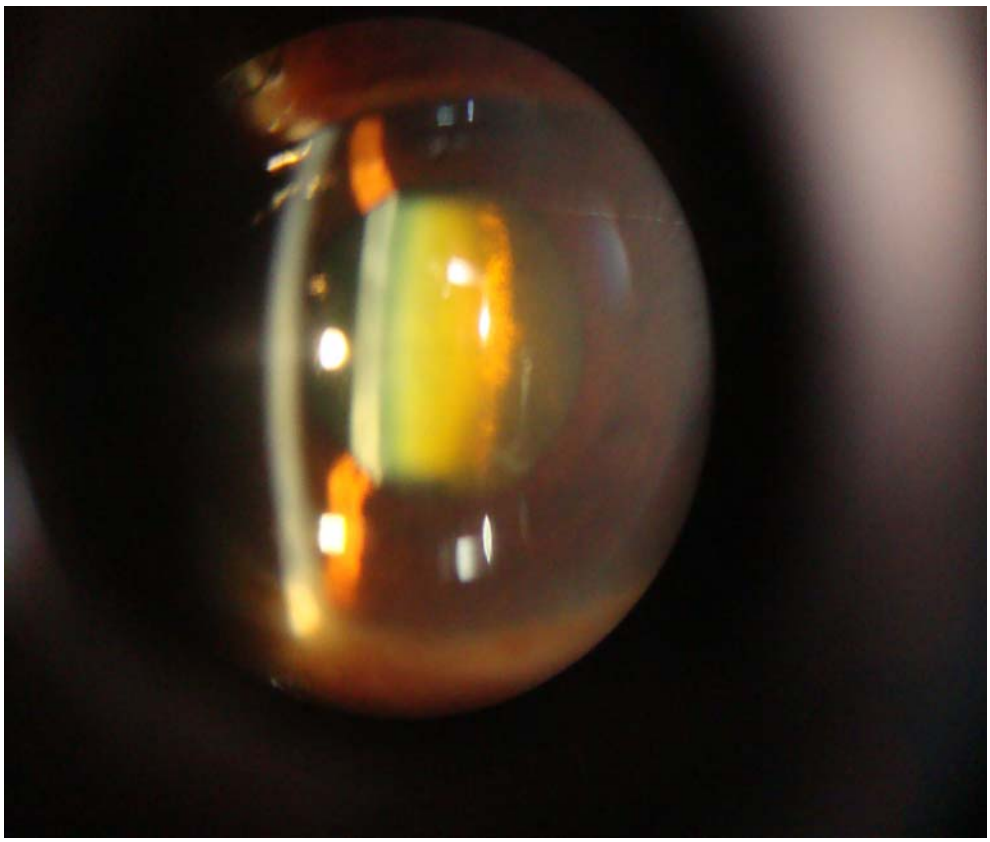
S No.	Name	Age	Sex	IP No	Associated systemic disease	Eye	Vision	Other eye vision	Pre op DR status RE	Pre op DR status LE	Post op DR status RE	Post op DR status LE	Final Visual acuity
75	Joseph	72	M	32871	HT	LE	6/60	6/60	MILD	MILD	MILD	M0D	6/12
76	Premkumari	43	F	34039		LE	6/24	6/12	NO	NO	NO	NO	6/12
77	Paramasivam	65	M	34062		RE	6/60	6/36	MILD	MILD	M0D	M0D	6/12
78	Venkatesan	67	M	35812	HT	LE	5/60	6/60	M0D	M0D	M0D	SEVERE	6/60
79	Selvaraj	45	M	36423		RE	6/36	6/18	NO	NO	NO	NO	6/12
80	MadinaBeevi	61	F	36780		LE	6/36	6/18	NO	NO	NO	MILD	6/12
81	Sivakumar	60	M	5756		RE	6/60	6/36	MILD	MILD	M0D	M0D	6/12
82	Murugesan	66	M	6732	HT	LE	5/60	6/60	M0D	M0D	M0D	M0D	6/12
83	Selvaperumal	50	M	11064		RE	6/36	6/18	NO	NO	NO	NO	6/18
84	Maheshwari	55	F	45929		LE	6/36	6/18	NO	NO	NO	MILD	6/12

LIST OF SURGERIES PERFORMED

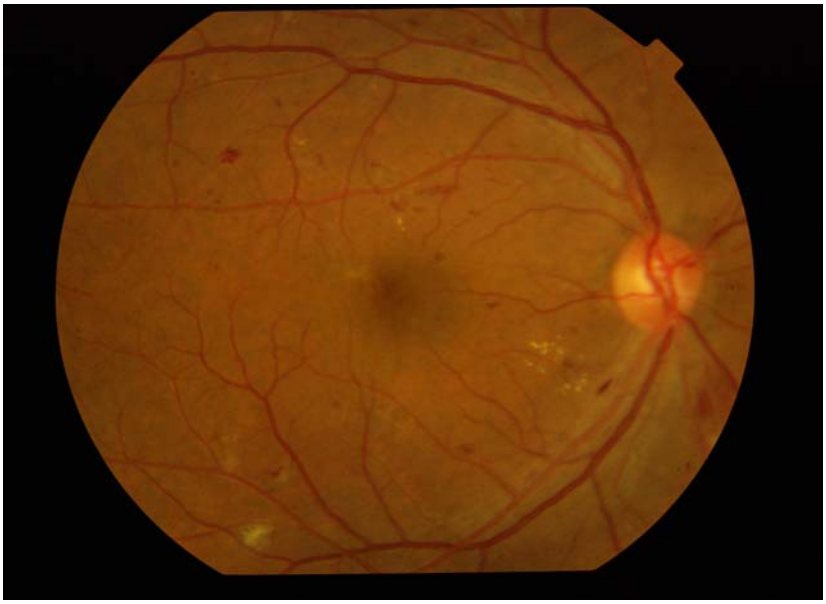
Sl. No.	Name	Age	Sex	Hosp. No.	Diagnosis	Surgery
1	Devaraj	55	M	3215	RE Mature cataract	ECCE with PCIOL
2	Noorjahan	60	F	9923	LE Mature cataract	ECCE with PCIOL
3	Rajammal	65	F	10124	LE Mature cataract	ECCE with PCIOL
4	Venkatesan	50	M	10774	RE Panophthalmitis with anterior staphyloma	Evisceration
5	Saroja	60	F	11552	RE Phacophormic glaucoma	ECCE with PCIOL
6	Jeyaraman	60	M	13040	RE Chronic dacryocystitis	Dacryocystectomy
7	Arumugam	60	M	13209	RE Mature cataract	ECCE with PCIOL
8	Kasthuri	22	F	178332	LE Lower lid Chalazion	Incision and curettage
9	Devammal	54	F	16289	RE Immature cataract	ECCE with PCIOL
10	Rani	68	F	14628	LE Immature cataract	ECCE with PCIOL
11	Mohanambal	70	F	14560	RE Mature cataract	ECCE with ACIOL
12	Muthu	40	M	14645	RE Nasal pterygium	Pterygium excision with autograft
13	Thangamalai	62	M	14668	RE Nuclear sclerosis	ECCE with PCIOL
14	Vasudeiah	60	M	22402	RE Immature cataract	SICS with PCIOL
15	Lakshmi	65	F	180554	RE Nuclear sclerosis	SICS with PCIOL
16	Kadarkalaiselvan	60	M	23699	LE Phacomorphic glaucoma	ECCE with PCIOL with trabeculectomy
17	Shanmugam	73	M	28301	LE Lmmature cataract	SICS with ACIOL

Sl. No.	Name	Age	Sex	Hosp. No.	Diagnosis	Surgery
18	Nagappan	70	M	29244	LE Mature cataract	SICS with PCIOL
19	Lakshmi	55	F	10493	LE Lagophthalmos	Tarsorrhaphy
20	Jeevarathinam	65	F	40765	RE Nuclear sclerosis	SICS with PCIOL

POSTERIOR SUBCAPSULAR CATARACT



PRE OPERATIVE MODERATE NON PROLIFERATIVE DIABETIC RETINOPATHY (OPERATED EYE)

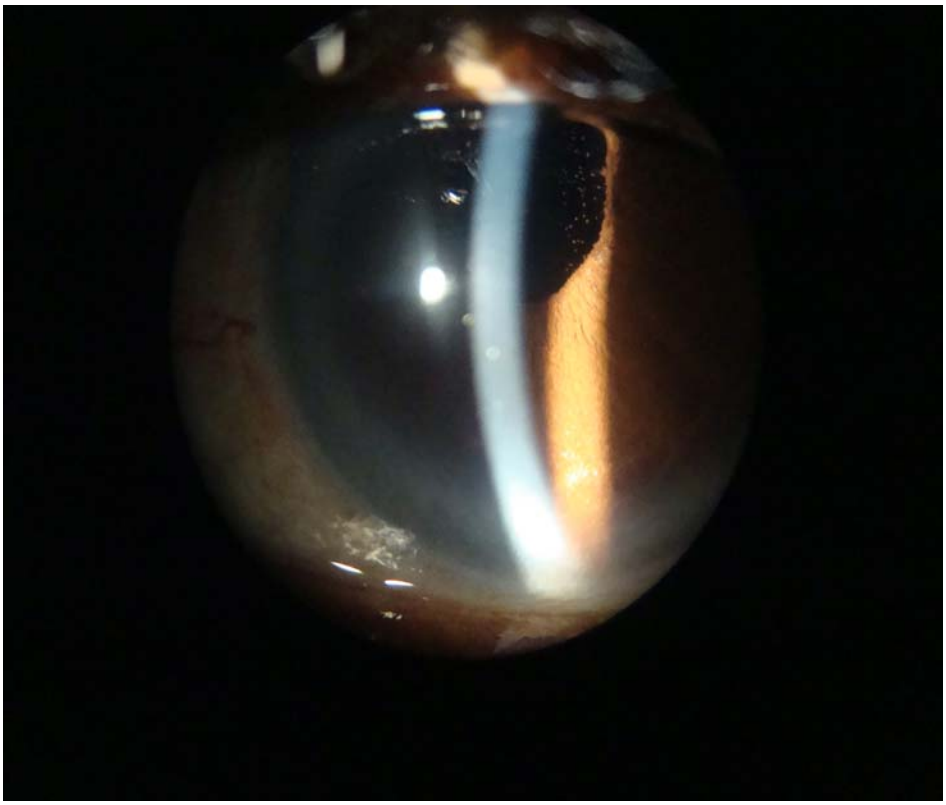


PROGRESSED TO SEVERE NON PROLIFERATIVE DIABETIC RETINOPATHY AFTER SURGERY

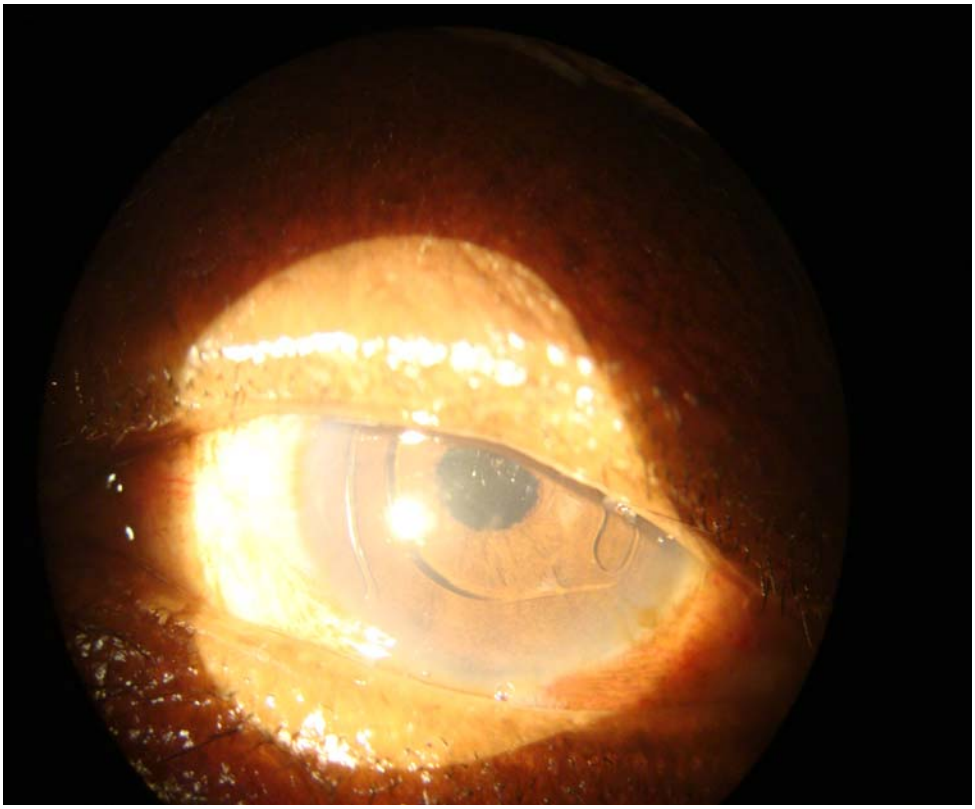
PRE OPERATIVE MODERATE NON PROLIFERATIVE DIABETIC RETINOPATHY (NON OPERATED EYE)

PROGRESSED TO SEVERE NON PROLIFERATIVE DIABETIC RETINOPATHY AFTER SURGERY (NON OPERATED EYE)

NORMAL POST OPERATIVE EYE



ANTERIOR CHAMBER INTRA OCULAR LENS IMPLANTATION DUE TO POSTERIOR CAPSULAR TEAR

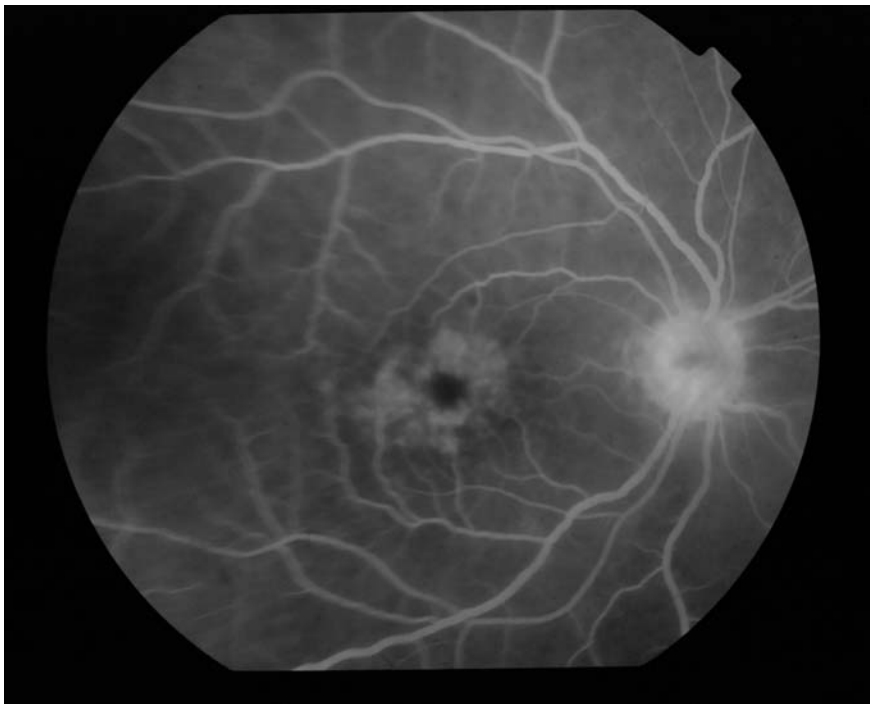


CLINICALLY SIGNIFICANT MACULAR EDEMA

FUNDUS FLUORESCEIN ANGIOGRAPHY OF NON PROLIFERATIVE DIABETIC RETINOPATHY WITH MACULAR EDEMA

CYSTOID MACULAR EDEMA

FUNDUS FLUORESCEIN ANGIOGRAPHY OF CYSTOID MACULAR EDEMA



STRIATE KERATOPATHY